UNITED STATES DISTRICT COURT

DISTRICT OF MAINE

DERMOT HARVEY, et al.,	***)***	
	)	
Plaintiffs	***)***	
	)	
v.	***)***	***Docket No. 98-85-P-DMC***
	)	
JAMES P. RINES, M.D., et al.,	***)***	
	)	
Defendants	***)***	

MEMORANDUM DECISION ON DEFENDANT MID-COAST HOSPITAL'S MOTION* IN LIMINE *ON CAUSATION TESTIMONY OF PLAINTIFFS' EXPERT WITNESSES AND PLAINTIFFS' MOTION* IN LIMINE *TO LIMIT THE SCOPE OF TESTIMONY OF THOMAS BROWNE, M.D., AND HENRY SPILLER, R.N., AND TO EXCLUDE TESTIMONY OF PHILIP GUZELIAN, M.D.

Mid-Coast Hospital, the remaining defendant in this medical malpractice action, has filed a motion *in limine* (Docket No. 25) asking the court to exclude or limit the testimony of the plaintiffs' expert witnesses as to causation. The plaintiffs, in turn, have filed a motion *in limine* (Docket No. 26) to exclude any and all testimony of one of the defendant's identified expert witnesses, Philip Guzelian, M.D., and to exclude certain opinion testimony given by the defendant's other expert witnesses at their depositions. I grant the plaintiffs' motion as to Dr. Guzelian and otherwise deny both motions.

I. Applicable Legal Standard

Evaluation of challenges to expert opinion testimony begins with Fed. R. Evid. 702 and

Daubert v. Merrell Dow Pharms., Inc., 509 U.S. 579 (1993). Rule 702 provides:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise.

In *Daubert*, the Supreme Court held that Rule 702 displaced earlier case law standards governing the admissibility of expert opinion testimony. 509 U.S. at 589.

> The *Daubert* Court's interpretation of Rule 702, drawn from its text, requires the trial judge to evaluate an expert's proposed testimony for both reliability and relevance prior to admitting it. The requisite review for reliability includes consideration of several factors: the verifiability of the expert's theory or technique, the error rate inherent therein, whether the theory or technique has been published and/or subjected to peer review, and its level of acceptance within the scientific community. The Court reasoned that due investigation of such matters will ensure that proposed expert testimony imparts "scientific knowledge" rather than guesswork. Withal, the factors that the Court enumerated do not function as a "definitive checklist or test," but form the basis for a flexible inquiry into the overall reliability of a proffered expert's methodology.

Ruiz-Troche v. Pepsi Cola of Puerto Rico Bottling Co., 161 F.3d 77, 80-81 (1st Cir. 1998) (citations omitted). Expert testimony must be relevant "in the incremental sense that the expert's proposed opinion, if admitted, likely would assist the trier of fact to understand or determine a fact in issue," *id*. at 81, or, in other words, Rule 702 "requires a valid scientific connection to the pertinent inquiry as a precondition to admissibility," *Daubert*, 509 U.S. at 592. The trial court must focus on the expert's methodology rather than on his or her conclusions. "A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered." *General Elec. Co. v. Joiner*, 118 S.Ct. 512, 519 (1997). "[T]rial judges may evaluate the data offered to support an expert's bottom-line opinions to determine if that data provides adequate support to mark the expert's testimony as reliable." *Ruiz-Troche*, 161 F.3d at 81.

II. The Plaintiffs' Experts

The defendant's motion does not differentiate among the three expert witnesses offered by the plaintiffs in urging exclusion of their proposed testimony on causation. The defendant argues that all such testimony should be excluded because

> none of the Plaintiffs' expert witnesses have cited to or have located any medical journal article, scientific publication, information or data which concludes that the administration of Dilantin or Valium, individually or in combination, in the context of carbamazepine poisoning provides any therapeutic or beneficial effect on seizure or status epilepticus. They argue, without scientific evidence or basis, that these anticonvulsant medications are beneficial in treating seizures and status epilepticus in eplileptic [sic] patients and by extension argue that they are also beneficial in treating seizures and status in cases of exogenous toxins. There are no studies which support this theory.
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> Dr. Shannon and Dr. Rutecki's theories have not undergone rigorous testing and study, nor has this theory been the subject of peer reviewed publications, no known rate of error nor any standards or controls on how much of these anticonvulsants can be used to prevent the effects of Tegretol poisoning.
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> * * *
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> There is no generally accepted theory or analysis in the medical community of the effect of ingesting massive concentrations of Tegretol. Likewise, there is no generally accepted theory in the medical community explaining how Dilantin or Valium work under the circumstances of a massive Tegretol overdose. There is no accepted effective treatment for a Tegretol overdose.

Defendant's Motion in Limine on Causation Testimony of Plaintiffs' Expert Witnesses (Docket No. 25) at 7-8. The defendant provides no citations to the record to support these assertions and, in any event, they are based on too narrow a view of the record and of the *Daubert* standard.

The defendant's position is essentially that, in order to offer an opinion in this case, the plaintiffs' experts must be able to support that opinion with studies or other evidence specific to the drug that induced the *status epilepticus* in this case, rather than scientific evidence concerning the appropriate treatment for *status epilepticus* in general, or even for *status epilepticus* induced by drug

overdose in general. That is not what Rule 702 and *Daubert* require. *See Mendes-Silva v. United States*, 980 F.2d 1482, 1486 (D.C.Cir. 1993) (study exactly duplicating conditions at issue in case at hand not necessary in order for expert to express opinion on causation; applying stricter pre-*Daubert* standard); *Wilson v. Petroleum Wholesale, Inc.*, 904 F. Supp. 1188, 1190-91 (D. Colo. 1995) (lack of scientific studies on specific issue on which expert stated opinion was matter for cross-examination rather than exclusion of opinion testimony; applying *Daubert*). As the First Circuit noted in *United States v. Alzanki*, 54 F.3d 994, 1006 (1st Cir. 1995), the fact that proffered expert testimony is more generalized than it might possibly be "may temper its probative value to the factfinder" but does not necessarily negate its relevance entirely.

Here, the plaintiffs offer the *Poisindex* in effect at the relevant time (Exh. 1 to Defendant's Motion in Limine on Causation Testimony of Plaintiffs' Expert Witnesses ("Defendant's Motion") (Docket No. 25)), which they assert without citation to authority "is the major primary reference used by poison control centers across the country to advise treating doctors how to treat specific types of overdoses." Plaintiffs' Objection to Defendant's Motion to Exclude Testimony of Doctors Shannon, Rutecki and Hodgdon ("Plaintiffs' Objection") (Docket No. 31) at 6. That document does direct that, when poisoning has been caused by carbamazepine, the generic name for Tegretol, and seizures result, diazepam (Valium) should be administered, followed by phenytoin (Dilantin) or phenobarbital if the seizures cannot be controlled or recur. *Poisindex*, Carbamazepine, at 0.4.2.E. They also offer: (1) an article by Daniel H. Lowenstein, M.D., and Brian K. Alldredge, Pharm. D., published in *The New England Journal of Medicine* in April 1998 in which drug toxicity is included as a cause of *status epilepticus* and treatment with diazepam and phenytoin is recommended, without any exception for *status epilepticus* induced by any particular drug (Exh. 3 to Plaintiffs' Motion *in*

Limine to Limit the Scope of Testimony of Thomas Browne, M.D. and Henry Spiller, R.N. and to Exclude Testimony of Philip Guzelian, M.D. ("Plaintiffs' Motion") (Docket No. 26)); (2) an article by David M. Treiman, M.D., and others published in *The New England Journal of Medicine* in September 1998 concerning a study of treatment of *status epilepticus* by various drug therapies, including diazepam and phenytoin, in which Dr. Rutecki was one of the investigators (Exh. 4 to Plaintiffs' Motion); (3) excerpts from *Handbook of Epilepsy*, written by Dr. Browne, the defendant's expert witness, and Gregory L. Holmes, specifically a chapter entitled "Status Epilepticus" in which the authors note that drug intoxication is a cause of *status epilepticus* and recommend diazepam and lorazepam as "initial drugs of choice in patients who are actively seizing," Exh. 5 to Plaintiffs' Motion at 224, without any restriction for patients whose seizures have been caused by carbamazepine overdose; (4) a chapter on *status epilepticus* written by Dr. Rutecki and included in a textbook published in 1992 which lists drug intoxication as a known cause of the condition and recommends use of diazepam or lorazepam to stop the convulsions (Exh. 6 to Plaintiffs' Motion); (5) a chapter on *status epilepticus* included in a 1991 textbook which includes drug toxicity as a cause and recommends therapy with diazepam or lorazepam and phenytoin (Exh. 7 to Plaintiffs' Motion); and (6) a chapter on *status epilepticus* and serial seizures from another textbook that recommends prompt drug therapy even in patients with seizures caused by drug overdose (Exh. 8 to Plaintiffs' Motion). The defendant has offered no studies or medical literature that dispute these sources with respect to carbamazepine overdose alone.

It is significant in this case that there is no offered written authority contradicting the position of the plaintiffs' experts and that none of them has admitted, or has even been asked to admit at deposition, that the information upon which they relied in forming their opinions in this case was not

of a type reasonably relied on by experts in the field. *Ambrosini v. Labarraque*, 101 F.3d 129, 138 (D.C.Cir. 1996); *Mendes-Silva*, 980 F.2d at 1487. On balance, the proposed testimony of the plaintiffs' experts will impart scientific knowledge rather than guesswork, based on their expressed methodology, and it will likely assist the trier of fact to understand or determine a fact in issue, thus meeting both the reliability and the relevance tests of *Daubert*. *Ruiz-Troche*, 161 F.3d at 81. Nothing further is required.

Accordingly, the defendant's motion to exclude or limit the testimony of Drs. Shannon, Rutecki and Hodgman for the plaintiffs is denied, subject to the right of the defendant to raise specific objections at trial to particular questions addressed to each of these expert witnesses or answers provided by them.

III. The Defendant's Experts

A. Dr. Browne

The plaintiffs seek an order excluding from trial certain opinions of Thomas Reed Browne, M.D., a neurologist, stated during his deposition, arguing that Dr. Browne is not qualified to express those opinions because he has not studied *status epilepticus* in the context of a Tegretol overdose, and that the challenged opinions are not reliable because there are no studies or medical literature that support them. Memorandum of Law in Support of Plaintiffs' Motion (Docket No. 26) at 8-10. As was the case with the defendant's motion *in limine*, this argument applies the *Daubert* standard too narrowly. *See, e.g., Lakie v. SmithKline Beecham*, 965 F. Supp. 49, 56 (D.D.C. 1997) (absence of studies not dispositive so long as methodology employed by expert is sound).

Dr. Browne opined at deposition that Julian Harvey, the patient at issue in this case, was not

in *status epilepticus* when the relevant seizures occurred and that standard drug treatment for *status epilepticus* was not indicated in this patient and would have created an additional danger of hypotension, or increasingly low blood pressure. Deposition of Thomas Reid [sic] Browne, III, M.D., ("Browne Dep.") (attached to Plaintiffs' Motion), at 49-51, 57-58, 74-76.

Both sides appear to agree that *status epilepticus*, or seizures of any kind, induced by Tegretol overdose, are very rare. The only information in the record concerning *status epilepticus* induced by overdose of any drugs indicates that it occurs in no more than 10% of all cases. Exh. 4 to Plaintiffs' Motion, Table 2 (5.2 - 6.3%); Exh. 6 to Plaintiffs' Motion at 1478 (10%). It is not surprising, therefore, that there are few if any studies specific to treatment of Tegretol-induced seizures, however defined. While Dr. Browne has never treated a patient with *status epilepticus* induced by Tegretol overdose, Browne Dep. at 58-59, he has published numerous papers concerning the use of anti-convulsive medication, including diazepam and phenytoin, in the treatment of *status epilepticus*, Curriculum Vitae of Thomas Reed Browne, III, M.D., Attachment 1 to Defendant's Memorandum in Opposition to Plaintiff's [sic] Motion in Limine to Limit the Testimony of Thomas Reed Browne, III, M.D., etc. ("Defendant's Opposition") (Docket No. 34)] at 5-17.

Dr. Browne's opinion that *status epilepticus* resulting from Tegretol overdose must be treated differently from *status epilepticus* resulting from any other cause, upon which the plaintiffs concentrate in their motion, if indeed that is his opinion, is not a necessary underpinning to his specific opinions that Julian Harvey was not in *status epilepticus* at the relevant time and that administration of Valium to Julian Harvey at the relevant time was contraindicated due to the brief nature of the seizures and his documented hypotension, which could be increased by the administration of Valium. Browne Dep. at 57-58, 74-76. It is, after all, the specific treatment

received or not received by Julian Harvey that is at issue in this case. In addition, the medical literature submitted by the defendant refers, albeit without direct comment, to cases in which seizures caused by carbamazepine overdose were not treated with anticonvulsant drugs. *See* Attachments 4 (Table 4), 5 (Table) to Defendant's Opposition.

The plaintiffs' arguments concerning Dr. Browne's proffered testimony are more applicable to its weight than to its admissibility. They do not accurately portray his opinion concerning *status epilepticus* in the context of an overdose of Tegretol, at least as presented in the pages of his deposition transcript to which they direct the court's attention. Plaintiffs' Motion at 7. In any event, the challenged opinions of Dr. Browne, as applied to the instant case, appear to me to be both reliable and relevant as those terms are used in *Daubert*. Accordingly, the motion to exclude specific portions of his proffered testimony will be denied, subject, of course, to the right of the plaintiffs to object to specific questions and answers at trial.

B. Henry Spiller, R.N.

The plaintiffs also seek to exclude certain statements of opinion by Henry Spiller, a registered nurse and clinical director of the Kentucky Regional Poison Center, Curriculum Vitae [of Henry Spiller] (Attachment 2 to Defendant's Opposition) at 1, who also proffered expert testimony on behalf of the defendant at deposition. The plaintiffs do not attack Spiller's qualifications but maintain that the challenged opinions are not reliable under *Daubert* because the rarity of *status epilepticus* in the context of a Tegretol overdose makes inadmissible any opinion that a treatment different from that appropriate for *status epilepticus* otherwise induced is appropriate, there is no medical literature supporting Spiller's opinions, and Spiller was uncertain about his opinion that a by-product of Tegretol injured Julian Harvey. Plaintiffs' Motion at 11-13.

The plaintiffs' first argument, carried to its logical extreme, would mean that medical professionals could not disagree in their testimony about the appropriate treatment for a rare medical condition because any differences in treatment between that condition and that considered appropriate for a more general category to which that condition might belong would be "untestable." To the contrary, so long as the facts differentiating that rare condition from the more general category are clearly stated by the expert witness, and the methodology by which that expert comes to his conclusion is set forth and verifiable, there is no reason to exclude the testimony merely because the patient presents with a rare condition. Contrary to the plaintiffs' argument, Judge Brody's unreported July 21, 1998 decision in *Coffin v. Orkin Exterminating Co.*, Docket No. 97-258-B, a copy of which is Attachment 2 to Plaintiffs' Motion, does not require a different result. In that case, Judge Brody excluded evidence of the plaintiff's claimed multiple chemical sensitivity ("MCS") on the ground that every federal court that had addressed the issue in a reported opinion (citing seven cases) had rejected expert testimony on MCS as too speculative. Order and Memorandum of Decision, *Coffin v. Orkin Exterminating Co.*, at 5-7. Nothing in Judge Brody's opinion suggests that it is the rarity of MCS that makes expert testimony about it inadmissible. In addition, it is again important to note that Spiller casts his opinion testimony in terms of Julian Harvey's condition at the relevant time, and any opinion that *status epilepticus* cannot be caused by a Tegretol overdose, if indeed that is Spiller's opinion, is not a necessary building block of his specific opinions concerning this case.

I have already discussed the plaintiffs' arguments concerning the lack of medical literature specifically addressing *status epilepticus* induced by a Tegretol overdose and will not repeat that discussion here. The plaintiffs' argument that "all literature and opinions . . . are to the contrary" to Spiller's opinion (Deposition of Henry Spiller, M.S., attached to Plaintiffs' Motion, at 99-100) that

Dilantin is an inappropriate treatment for recurrent seizures resulting from a Tegretol overdose, Plaintiffs' Motion at 12, is incorrect. Case reports in which Dilantin was not used, without comment, as noted above, cannot be said to be contrary to Spiller's opinion.

The plaintiffs assert that Spiller opined "[t]hat Julian Harvey was injured from the effects of a Tegretol by-product, 10-11 Epoxide." Plaintiffs' Motion at 11. They cite page 85 of the transcript of his deposition. At that page, when asked why an overdose of Tegretol with a serum level of 37 to 40 was "overwhelming," Spiller responded:

> There is another portion to carbamazepine overdose. There's a metabolite called 10,11-epoxide carbamazepine that was not measured in this case . . . that is profoundly toxic. And that may play a great role in these cases that — of apparent severe outcomes with serum levels similar to those that did not have severe outcomes.

Spiller Dep. at 85-86. Spiller then agreed that the level of 10,11-epoxide was not measured in Julian Harvey, so "[n]o one would know" whether it played a role in this case. *Id*. at 86. The defendant does not address this aspect of the plaintiffs' motion in its opposition. I do not read Spiller's quoted testimony to assert that Julian Harvey was injured by 10,11-epoxide carbamazepine. When and if he offers such an opinion and an objection is made, I will rule on it. That is not the situation at this time. Counsel should bear in mind, however, the discussion of Dr. Guzelian's proffered testimony which follows before offering such testimony from Spiller.

C. Philip S. Guzelian, Jr., M.D.

The plaintiffs attack both the qualifications of Dr. Guzelian and the reliability of his proffered opinions, and they seek to exclude any testimony from him. The plaintiffs contend that Dr. Guzelian, a board-certified internist and liver specialist, Deposition of Philip S. Guzelian, Jr., M.D. ("Guzelian Dep.") (attached to Plaintiffs' Motion) at 35, has no direct experience or scientific

knowledge that qualify him to opine about this case, that his opinions were formulated specifically for this litigation, and that his opinions lack a reliable foundation in fact, medical literature or his own experience. Plaintiffs' Motion at 13-17. The defendant responds, without citation to authority or the record, that

> Dr. Guzelian is being proffered as an expert witness to testify concerning the metabolic processes which effect [sic] the body's response to Tegretol and he has extensive experience in the enzyme P-450 3A4 which processes the active ingredients of Carbamazepine. He has extensive knowledge of the metabolism of Tegretol. He is a general toxicologist by training, education and experience, he has performed research and written extensive publications in the field of toxicology.

Defendant's Opposition at 16.

Dr. Guzelian's extensive list of publications does not include any that mention carbamazepine or seizures induced by drug overdose in their titles. Curriculum Vitae [of Philip S. Guzelian, M.D.] (Attachment 3 to Defendant's Opposition) at 8-27. He testified that he had never treated any Tegretol overdose patients, Guzelian Dep. at 32; he has "substantial knowledge of the metabolism of Tegretol," but that his experience with the outcome of Tegretol overdose was drawn from a review of literature rather than personal experience, *id*. at 33; he does not treat *status epilepticus*, and that his opinions regarding *status epilepticus* in this case are based on a review of the medical literature that he conducted for this case, *id*. at 34; he has done no clinical studies on overdose patients, *id*. at 41-42; he spends 10% of his time in patient care and about 65% in research, *id*. at 43; he does not claim to be an expert in the treatment of *status epilepticus*, *id*. at 65; if he were treating a patient in *status epilepticus* in the context of a carbamazepine overdose he "may or may not" use Dilantin or Valium, *id*. at 70-71; it is "very clear" that there is no scientific basis "supporting the conclusion that treatment with anticonvulsants is known to be a safe and effective means of dealing with *status*

epilepticus caused by carbamazepine," based on his review of the medical literature, *id*. at 71-72; "in fact there are some indications that [treatment of carbamazepine-induced *status epilepticus* with anticonvulsants] is [harmful]— or it might have had no effect whatsoever. So my opinion is you just don't know," *id*. at 73; he assumed that carbamazepine-10,11-epoxide was formed in Julian Harvey and that it was toxic, *id*. at 79; "from [his] knowledge of the metabolism and drug interactions potentially involved in carbamazepine metabolism and from some case reports that [he has] reviewed, there is good evidence —or there is some evidence to suggest that, in fact, Dilantin could actually be detrimental," *id*. at 92; and he did not say that treatment of Julian Harvey with Dilantin would not have been appropriate, *id*. at 95, but someone who chose not to treat with Dilantin "would be on solid scientific ground," *id*. at 96.

The defendant focuses on Dr. Guzelian's deposition testimony that Dilantin "could actually be detrimental" to a patient suffering from carbamazepine overdose:

> The reason is that carbamazepine is oxidized to the 10,11-epoxide by cytochrome P4503A4 in the liver, potentially in other tissues, but probably in the liver. Now, there is evidence — and you cited a couple of the papers earlier — to indicate that the 10,11-epoxide is at least as toxic, and some people believe is actually more neurotoxic than is the parent drug, carbamazepine, itself.
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> Dilantin has the effect of increasing the amount of cytochrome P4503A4 in the liver. Since the amount of cytochrome P4503A4 in the liver — it may indeed be limiting for the formation of the 10,11 carbamazepine epoxide. The effect of Dilantin would actually be to convert the carbamazepine to a potentially more toxic derivative. It therefore would actually enhance the potential to cause seizures or other anticholinergic effects, but particularly in this case we're talking about neurotoxicity, so seizures.
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> And in fact, I cited for you a couple of papers in which they report patients who are already on Dilantin, already have a therapeutic does of Dilantin on board, in which they added carbamazepine and actually made seizures worse, and then they stopped carbamazepine, and the seizures go away.
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> So all of that evidence — mechanistic and clinical evidence would

> coalesce into a coherent argument that in fact Dilantin may not only not be beneficial, but it may actually make toxicity — neurotoxicity of carbamazepine worse.
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> [W]e've never found a human being who doesn't have cytochrome P4503A4 in the liver. So it's reasonable to presume that every patient who gets one of these overdoses has this in the liver. Therefore, it would be — it would really be logical to assume, even though it's not measured, that every patient who gets carbamazepine metabolizes to some extent the carbamazepine to the 10,11-epoxide. We also know that the 10,11-epoxide is at least as toxic, if not more toxic than the parent drug.
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> That's the indication from the literature.

Id. at 92-93, 100-01.

I have several problems with Dr. Guzelian's proffered testimony. First, he and the defendant offer no basis for his claimed familiarity with the metabolism of carbamazepine, nor for his assumption that seizures would be made worse by the presence of 10,11-epoxide of carbamazepine. Second, he draws his conclusions from a lack of published literature to support the position that carbamazepine-induced *status epilepticus* may be effectively treated with Valium and Dilantin, an analytical practice that I have declined to apply with respect to the proffered testimony of other experts in this case. Third, Dr. Guzelian rests his opinion on certain presumptions about the level of carbamazepine and 10,11-epoxide of carbamazepine in Julian Harvey. *See Mancuso v. Consolidated Edison Co. of New York, Inc.*, 967 F.Supp. 1437, 1450-51 (S.D.N.Y. 1997) (improper for expert to presume that plaintiff must have been exposed to sufficiently high dose of toxin). Finally, and most important, Dr. Guzelian's deposition testimony, taken as a whole, clearly reveals that his proffered opinions are based primarily upon a review of certain medical literature undertaken specifically for this case. *See Wade-Greaux v. Whitehall Labs., Inc.*, 874 F. Supp. 1441, 1476 (D.V.I. 1994) (physician who had not engaged in any studies relating to issue upon which he offers expert

opinion and whose only knowledge or experience comes from review of selected literature for purpose of testifying is not qualified to offer opinion testimony). *See generally Mancuso*, 967 F. Supp. at 1442-54 (finding proffered expert insufficiently qualified). In addition, Dr. Guzelian, a researcher who spends only 10% of his time in clinical practice, provides nothing in his deposition testimony to support a conclusion that the methodology he used to reach his conclusions in this case is one that would be accepted by medical professionals treating a patient with seizures due to carbamazepine overdose, which is the group of individuals against whom his testimony is proffered. On the record presented, I am left with the impression that Dr. Guzelian's proffered testimony is more "unscientific speculation offered by a genuine scientist" than it is "genuinely scientific," to use the description of the *Daubert* distinction penned by Judge Posner. *Rosen v. Ciba-Geigy Corp*., 78 F.3d 316, 318 (7th Cir. 1996). Accordingly, the motion to exclude Dr. Guzelian's testimony is granted.

IV. Conclusion

For the foregoing reasons, the defendant's motion in limine to limit or exclude the testimony of Drs. Shannon, Rutecki, and Hodgman is **DENIED**. The plaintiffs' motion to limit or exclude the testimony of certain expert witnesses proffered by the defendant is **GRANTED** as to Dr. Guzelian and otherwise **DENIED**.

Dated this 19th day of January, 1999.

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David M. Cohen
United States Magistrate Judge